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DATE: Sunday, June 30, 2002 Printable Copy Create Case

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☐ 1. Document ID: US 6407133 B2

L1: Entry 1 of 7 File: USPT Jun 18, 2002

US-PAT-NO: 6407133
DOCUMENT-IDENTIFIER: US 6407133 B2

TITLE: Treatment and prevention of reactive oxygen metabolite-mediated cellular damage

DATE-ISSUED: June 18, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Hellstrand; Kristoffer	Gothenburg			SEX
Hermodsson; Svante	Molndal			SEX
Gehlsen; Kurt R.	Encinitas	CA		

US-CL-CURRENT: 514/400

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 2. Document ID: US 6242473 B1

L1: Entry 2 of 7 File: USPT Jun 5, 2001

US-PAT-NO: 6242473
DOCUMENT-IDENTIFIER: US 6242473 B1

TITLE: Treatment and prevention of reactive oxygen metabolite-mediated cellular damage

DATE-ISSUED: June 5, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Hellstrand; Kristoffer	Goteborg			SEX
Hermodsson; Svante	Molndal			SEX
Gehlsen; Kurt R.	Encinitas	CA		

US-CL-CURRENT: 514/400

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 3. Document ID: US 6203997 B1

L1: Entry 3 of 7 File: USPT Mar 20, 2001

US-PAT-NO: 6203997
DOCUMENT-IDENTIFIER: US 6203997 B1

TITLE: Quantitation of analytes in whole blood

DATE-ISSUED: March 20, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Romaschin; Alexander D.	Etobicoke			CAX
Walker; Paul M.	Toronto			CAX

US-CL-CURRENT: 435/7.2; 435/24, 435/34, 435/5, 435/7.1, 435/7.24, 435/7.31, 435/7.32, 435/962, 435/968, 435/975, 436/513 , 436/518, 436/536, 436/539

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☐ 4. Document ID: US 6190872 B1

L1: Entry 4 of 7 File: USPT Feb 20, 2001

US-PAT-NO: 6190872
DOCUMENT-IDENTIFIER: US 6190872 B1

TITLE: Method for identifying and monitoring patients at risk for systemic inflammatory conditions and apparatus for use in this method

DATE-ISSUED: February 20, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Slotman; Gus J.	Moorestown	NJ	08057	

US-CL-CURRENT: 435/7.92; 435/34, 435/69.4, 435/7.24, 435/7.32, 435/7.4, 514/12, 514/18, 514/2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☐ 5. Document ID: US 6077665 A

L1: Entry 5 of 7 File: USPT Jun 20, 2000

US-PAT-NO: 6077665
DOCUMENT-IDENTIFIER: US 6077665 A

TITLE: Rapid assay for infection in neonates

DATE-ISSUED: June 20, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Weirich; Erica E.	Redwood City	CA		
Rabin; Ronald L.	Rockville	MD		
Maldonado; Yvonne	Redwood City	CA		
Benitz; William E.	Palo Alto	CA		
Herzenberg; Leonore A.	Stanford	CA		
Herzenberg; Leonard A.	Stanford	CA		

US-CL-CURRENT: 435/6; 435/7.21

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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☐ 6. Document ID: US 5885781 A

L1: Entry 6 of 7 File: USPT Mar 23, 1999

US-PAT-NO: 5885781
DOCUMENT-IDENTIFIER: US 5885781 A

TITLE: Regulation of cytokine synthesis and release

DATE-ISSUED: March 23, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Johnson; Kirk	Moraga	CA		
Creasey; Abla A.	Piedmont	CA		
Aarden; Lucien A.	Amsterdam			NLX

US-CL-CURRENT: 435/7.1; 424/278.1, 436/63, 514/2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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☐ 7. Document ID: US 5389522 A

L1: Entry 7 of 7 File: USPT Feb 14, 1995

US-PAT-NO: 5389522
DOCUMENT-IDENTIFIER: US 5389522 A

TITLE: Serum antioxidants as predictors of the adult respiratory distress syndrome in septic patients

DATE-ISSUED: February 14, 1995

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Repine; John E.	Englewood	CO	80110	
Leff; Jonathan A.	Littleton	CO	80121	

US-CL-CURRENT: 435/7.4; 435/25, 435/27, 435/7.9, 435/7.92, 435/7.95

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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Term	Documents
"INTERLEUKIN 8".USPT.	0
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L1: Entry 3 of 7 File: USPT Mar 20, 2001

DOCUMENT-IDENTIFIER: US 6203997 B1
TITLE: Quantitation of analytes in whole blood

Brief Summary Paragraph Right (3):
"Sepsis" is defined as a pathological condition of the body resulting from the presence of infectious microorganisms, which clinically manifests as one or more of the following sequelae: pyrexia, hypotension, hypoxemia, tachycardia, hypothermia, neutrophilia, and neutropenia.

Brief Summary Paragraph Right (19):
The preselected analyte may be selected from any of a number of substances, proteins, and other macromolecules present in blood, such as infectious microorganisms, their toxic products, inflammatory mediators, hormones, acute phase proteins, toxins, drugs of abuse, markers of cardiac muscle damage, therapeutic drugs, cytokines, chemokines, etc. For example, the extent of sepsis or stage of infection in a human or animal patient may be determined by quantitating sepsis-associated markers, such as antigens of Gram-negative bacteria, Gram-positive bacteria, viruses, fungi, or inflammatory mediators such as tumor necrosis factor (TNF), interleukin-1. interleukin-6, interleukin-8 (IL-8). interferons and transforming growth factor .beta. (TGF-.beta.). Hormones may include thyroid hormones and human chorionic gonadotropin. Therapeutic drugs may include digoxin and theophylline. Drugs of abuse may include heroin and cocaine. Markers of cardiac damage may include myoglobin, troponin, and myosin light chain.

CLAIMS:

10. The method of claim 9 wherein said inflammatory mediator is selected from the group consisting of tumor necrosis factor, interleukin-1, interleukin-6, interleukin-8, interferon, and transforming growth factor .beta..

14. The diagnostic kit of claim 13 wherein said inflammatory mediator is selected from the group consisting of tumor necrosis factor, interleukin-1, interleukin-6, interleukin-8, interferon, and transforming growth factor .beta..

L1: Entry 4 of 7 File: USPT Feb 20, 2001

DOCUMENT-IDENTIFIER: US 6190872 B1
TITLE: Method for identifying and monitoring patients at risk for systemic inflammatory conditions and apparatus for use in this method

Brief Summary Paragraph Right (1):
Physiologic insults triggering the onset of systemic inflammatory conditions including sepsis, Adult Respiratory Distress Syndrome (ARDS), Systemic Inflammatory Response Syndrome (SIRS) and Multiple Organ Dysfunction Syndrome (MODS) have been identified to include infection and its systemic effects, shock, trauma, inhalation injury, pancreatitis, hypertransfusion, drug overdose, and near-drowning among others. The host response manifested in each of these insults includes increased capillary permeability, organ failure, and death. The mechanism of the response involves diffuse pathologic activation of inflammatory mediators including, but not limited to, endotoxin, leukotrienes B.sub.4, C.sub.4, D.sub.4 and E.sub.4, prostacyclin and thromboxane A.sub.2, activated granulocytes and complement components C3a and C5a, tumor necrosis factor, interleukin-1, interleukin-6, interleukin-8, and other cytokines, neutrophil elastase, platelet activating factor, nitric oxide, and oxide radicals.

Detailed Description Paragraph Right (2):
It is believed that systemic inflammatory conditions, particularly ARDS, SIRS and MODS, are the result of a severe generalized autodestructive inflammation. ARES is manifested clinically by hypoxemia, hypocapnia, diffuse infiltrates on chest roentgenogram and normal or low left ventricular filling pressures. Circulating prostaglandins, activated complement and abnormal intravascular aggregation of neutrophils have been implicated as possible mediators of ARDS. Slotman et al., Arch Surg. 121:271-274, 1986. Thromboxane B.sub.2 (TxB), prostaglandin 6-keto-F1.alpha. (PGI), activated complement components C3a and C5a, and granulocyte aggregation (GA) were found to be significantly elevated in all critically ill patients as compared to normal controls. For patients with ARDS the ratios of TxB/PGI and C3a/C5a also were significantly greater than controls. Differences between patients with and without ARDS in this study, however, were significant only for increased GA and plasma C3a in ARDS.

Detailed Description Paragraph Right (7):
In addition, a method is provided to monitor changes in selected physiologic parameters in patients to evaluate a treatment of a systemic inflammatory condition which comprises determining selected physiologic parameters of a patient; generating a SMART profile for the patient from the determined physiologic parameters; monitoring changes in selected physiologic parameters from said profile in a response to a treatment; and comparing the changes in the profile with an established control profile to monitor the treatment of patients at risk of developing a systemic inflammatory condition based on the comparison. For purposes of this invention, a "control profile" can either be generated from a data base containing mean values for the measured physiologic parameters from a population of patients with similar conditions and/or injuries or profiles of changing parameters associated with a similar condition and/or injury, or can be generated from the same patient to compare and monitor changes in the measured physiologic parameters over time. The SMART profile of the present invention is generated in the following manner. Selected physiologic parameters, which for the purposes of this invention includes at least the following physiologic parameters: physical examination, vital signs, hemodynamic measurements and calculations, clinical laboratory tests,

concentrations of acute inflammatory response mediators, and endotoxin levels, are determined in a patient. In a preferred embodiment, levels of prostaglandin 6-keto F1.alpha. (PGI) (the stable metabolite of prostacyclin), thromboxane B.sub.2 (TxB) (the stable metabolite of thromboxane A.sub.2), leukotrienes B.sub.4, C.sub.4, D.sub.4 and E.sub.4, interleukin-1.beta., interleukin-6, interleukin-8, tumor necrosis factor, neutrophil elastase, complement components C3 and C5a, platelet activating factor, nitric oxide metabolites and endotoxin levels are determined in a biological sample obtained from a patient at baseline and daily thereafter. As one of skill in the art will appreciate upon this disclosure, as other significant inflammatory response mediators are identified, they can also be measured and incorporated into the database as part of the SMART profile. Examples of biological samples include, but are not limited to, blood, plasma, serum, urine, bronchioalveolar lavage, sputum, and cerebrospinal fluid.

Detailed Description Paragraph Right (37):
The purpose of this study was to demonstrate the ability of the SMART method to identify interactions among physiologic parameters, standard hospital laboratory tests, patient demographics, and circulating cytokine levels that predict continuous and dichotomous dependent clinical variables in advance in individual patients with severe sepsis and septic shock. Patients (n=303) with severe sepsis or septic shock were entered into the placebo arm of a multi-institutional clinical trial. The patients were randomly divided into a model-building training cohort (n=200) and a prospective validation or predictive cohort (n=103). Demographics, including sex, race, age, admitting service (surgery or non-surgical), and co-morbidities were recorded at baseline for each patient. At baseline and on days 1 through 7, 14, 21, and 28, the physiologic parameters and hospital laboratory tests listed on Table 1 were recorded. In addition, at baseline and on days 1, 2, 3, and 4 plasma concentrations of interleukin-6 (IL-6), interleukin-8 (IL-8), and granulocyte colony stimulating factor (GCSF) were measured by ELISA using commercially available kits and standard ELISA methodology.

CLAIMS:

1. A method of identifying a patient at risk for developing a selected systemic inflammatory condition prior to development of signs and symptoms which are diagnostic of the selected systemic inflammatory condition comprising;

a) measuring selected physiological parameters in the patient, wherein measured selected physiologic parameters comprise physical examination, vital signs, hemodynamic measurements and calculations, clinical laboratory tests and concentrations of acute inflammatory response mediators, wherein said acute inflammatory response mediators comprise prostaglandin 6-keto F1.alpha., thromboxane B.sub.2, leukotrienes B.sub.4, C.sub.4, D.sub.4 and E.sub.4, interleukin 1.beta., interleukin-6, interleukin-8, tumor necrosis factor, neutrophil elastase, complements C3a and C5a, platelet activating factor, nitric oxide metabolites and endotoxin levels;

b) producing a systemic mediators-associated response test profile for the patient, wherein said systemic mediators-associated response test profile comprises the physiological parameters set forth in step (a); and

c) comparing said profile with an established control profile to identify the patient as being at risk for developing a selected systemic inflammatory condition prior to development of signs and symptoms which are diagnostic of the selected systemic inflammatory condition wherein the systemic inflammatory condition is selected from a group consisting of Adult Respiratory Distress Syndrome, Systemic Inflammatory Response Syndrome, sepsis, Multiple Organ Dysfunction Syndrome, single organ dysfunction, shock, transplant rejection, cancer and trauma.

2. A method of identifying a patient at risk for developing a selected systemic inflammatory condition prior to development of signs and symptoms which are diagnostic of the selected systemic inflammatory condition comprising:

a) measuring selected physiological parameters in the patient, wherein measured selected, physiologic parameters comprise physical examination, vital signs,

hemodynamic measurements and calculations, clinical laboratory tests and concentrations of acute inflammatory response mediators, wherein said acute inflammatory response mediators comprise prostaglandin 6-keto F1.alpha., thromboxane B.sub.2, leukotrienes B.sub.4, C.sub.4, D.sub.4 and E.sub.4, interleukin 1.beta., interleukin-6, interleukin-8, tumor necrosis factor, neutrophil elastase, complements C3a and C5a, platelet activating factor, nitric oxide metabolites and endotoxin levels;

b) producing a systemic mediators-associated response test profile for the patient, wherein said systemic mediators-associated response test profile comprises the physiological parameters as set forth in step (a); and

c) comparing said profile with an established control profile to identify the patient as being at risk for developing a selected systemic inflammatory condition prior to development of signs and symptoms which are diagnostic of the selected systemic inflammatory condition wherein the systemic inflammatory condition is selected from a group consisting of Adult Respiratory Distress Syndrome, Systemic Inflammatory Response Syndrome, sepsis, Multiple Organ Dysfunction Syndrome, single organ dysfunction, shock, transplant rejection, cancer and trauma, wherein concentrations at acute inflammatory response mediators are measured simultaneously via enzyme linked immunosorbent assays in a multiple analysis grid for grouped independent enzyme linked immunosorbent assays comprising:

an antibody grid;

a reagent grid placed on top of said antibody grid; and

an injection grid placed on top of said reagent grid.

3. A method of quantitatively predicting selected physiological parameters in a patient having or at risk for a selected systemic inflammatory condition comprising:

a) measuring selected physiological parameters in said patient, wherein measured selected physiologic parameters comprise physical examination, vital signs, hemodynamic measurements and calculations, clinical laboratory tests and concentrations of acute inflammatory response mediators, wherein said acute inflammatory response mediators comprise prostaglandin 6keto F1.alpha., thromboxane B.sub.2, leukotrienes B.sub.4, C.sub.4, D.sub.4 and E.sub.4, interleukin-1.beta., interleukin-6, interleukin-8, tumor necrosis factor, neutrophil elastase, complements C3a and C5a, platelet activating factor, nitric oxide metabolites and endotoxin levels;

b) producing a systemic mediators-associated response test profile for said patient, wherein said systemic mediators-associated response-test profile comprises the physiological parameters as set forth in step (a); and

c) comparing said profile with an established control profile from multiple patients with systemic inflammatory disease so that selected physiological parameters in said patient having or at risk for a selected systemic inflammatory condition are determined wherein the selected systemic inflammatory condition is selected from a group consisting of Adult Respiratory Distress Syndrome, Systemic Inflammatory Response Syndrome, sepsis, Multiple Organ Dysfunction Syndrome, single organ dysfunction, shock, transplant rejection, cancer and trauma.

4. A method of quantitatively predicting selected physiological parameters in a patient having or at risk for a selected systemic inflammatory condition comprising:

a) measuring selected physiological parameters in said patient, wherein measured selected physiologic parameters comprise physical examination, vital signs, hemodynamic measurements and calculations, clinical laboratory tests and concentrations of acute inflammatory response mediators, wherein said acute inflammatory response mediators comprise prostaglandin 6-keto F1.alpha., thromboxane B.sub.2, leukotrienes B.sub.4, C.sub.4, D.sub.4 and E.sub.4, interleukin 1.beta.,

interleukin-6, interleukin-8, tumor necrosis factor, neutrophil elastase, complements C3a and C5a, platelet activating factor, nitric oxide metabolites and endotoxin levels;

b) producing a systemic mediators-associated response test profile for said patient, wherein said systemic mediators-associated response test profile comprises the physiological parameters as set forth in step (a); and

c) comparing said profile with an established control profile from multiple patients with systemic inflammatory disease so that selected physiological parameters in said patient having or at risk for a selected systemic inflammatory condition are determined wherein the selected systemic inflammatory condition is selected from a group consisting of Adult Respiratory Distress Syndrome, Systemic Inflammatory Response Syndrome, sepsis, Multiple Organ Dysfunction Syndrome, single organ dysfunction, shock, transplant rejection, cancer and trauma, wherein concentrations of acute inflammatory response mediators are measured simultaneously via enzyme linked immunosorbent assays in a multiple analysis grid for grouped independent enzyme linked immunosorbent assays comprising;

an antibody grid;

a reagent grid placed on top of said antibody grid; and

an injection grid placed on top of said reagent grid.

5. A method of monitoring changes in selected physiological parameters in a patient to assess a treatment of a systemic inflammatory condition comprising:

a) measuring selected physiologic parameters in a patient, wherein measured selected physiologic parameters comprise physical examination, vital signs, hemodynamic measurements and calculations, clinical laboratory tests and concentrations of acute inflammatory response mediators, wherein said acute inflammatory response mediators comprise prostaglandin 6keto F1.alpha., thromboxane B.sub.2, leukotrienes B.sub.4, C.sub.4, D.sub.4 and E.sub.4, interleukin 1.beta., interleukin-6, interleukin-8, tumor necrosis factor, neutrophil elastase, complements C3a and C5a, platelet activating factor, nitric oxide metabolites and endotoxin levels;

b) producing a systemic mediators-associated response test profile for the patient, wherein said systemic mediators-associated response test profile comprises the physiological parameters as set forth in step (a);

c) monitoring changes in the measurements in said profile; and

d) comparing changes in said profile with an established control profile to monitor treatment of a systemic inflammatory condition wherein the systemic inflammatory condition is selected from a group consisting of Adult Respiratory Distress Syndrome, Systemic Inflammatory Response Syndrome, sepsis, Multiple Organ Dysfunction Syndrome, single organ dysfunction, shock, transplant rejection, cancer and trauma.

6. A method of Monitoring changes in selected physiological parameters in a patient to assess a treatment of a systemic inflammatory condition comprising:

a) measuring selected physiologic parameters in a patient, wherein measured selected physiologic parameters comprise physical examination, vital signs, hemodynamic measurements and calculations, clinical laboratory tests and concentrations of acute inflammatory response mediators, wherein said acute inflammatory response mediators comprise prostaglandin 6-keto F1.alpha., thromboxane B.sub.2, leukotrienes B.sub.4, C.sub.4, D.sub.4 and E.sub.4, interleukin 1.beta., interleukin-6, interleukin-8, tumor necrosis factor, neutrophil elastase, complements C3a and C5a, platelet activating factor, nitric oxide metabolites and endotoxin levels;

b) producing a systemic mediators-associated response test profile for the patient, wherein said systemic mediators-associated response test profile comprises the physiological parameters as set forth in step (a);

c) monitoring changes in the measurements in said profile; and

d) comparing changes in said profile with an established control profile to monitor treatment of a systemic inflammatory condition wherein the systemic inflammatory condition is selected from a group consisting of Adult Respiratory Distress Syndrome, Systemic Inflammatory Response Syndrome, sepsis, Multiple Organ Dysfunction Syndrome, single organ dysfunction, shock, transplant rejection, cancer and trauma, wherein concentrations of acute inflammatory response mediators are measured simultaneously via enzyme linked immunosorbent assays in a multiple analysis grid for grouped independent enzyme linked immunosorbent assays comprising wherein the enzyme linked immunosorbent assays are performed using a multiple analysis grid for grouped independent enzyme linked immunosorbent assays comprising:

(a) an antibody grid;

(b) a reagent grid placed on top of said antibody grid; and

(c) an injection grid placed on top of said reagent grid.